Appln # 09/744,384
Paper # 11 Attach.

********** Welcome to STN International **********

FILE 'HOME' ENTERED AT 15:51:08 ON 22 MAR 2002

FILE 'REGISTRY' ENTERED AT 15:51:38 ON 22 MAR 2002
L1 2 ASCORBIC ACID/CN
E ASCORBIC ACID/CN
L2 2 S E3

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE' ENTERED AT 16:07:19 ON 22 MAR 2002
L3 0 L-ASCORBIC ACID/CA
L4 13900 L-ASCORBIC ACID
L5 220 L4 (P) DIFFERENTIA?
L6 178 L4 (S) DIFFERENTIA?
L7 5 L6 (S) (NEURON OR CNS OR DOPAMIN?)
L8 3 DUP REM L7 (2 DUPLICATES REMOVED)

L1 ANSWER 2 OF 2 REGISTRY COPYRIGHT 2002 ACS
RN 50-81-7 REGISTRY
CN L-Ascorbic acid (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN (+)-Ascorbic acid
CN 3-keto-L-Gulofuranolactone
CN 3-Oxo-L-gulofuranolactone
CN Adenex
CN Allercorb
CN Antiscorbic vitamin
CN Antiscorbutic vitamin
CN Ascoltin
CN Ascorbajen
CN Ascorbic acid
CN Ascorbutina
CN Ascorin
CN Ascorteal
CN Ascorvit
CN C-Quin
CN C-Vimin
CN Cantan
CN Cantaxin
CN Catavin C
CN Ce-Mi-Lin
CN Ce-Vi-Sol
CN Cebicure
CN Cebion
CN Cebione
CN Cecon
CN Cegiolan
CN Ceglion
CN Celaskon
CN Celin

CN Cemagyl
CN Cenetone
CN Cereon
CN Cergona
CN Cescorbat
CN Cetamid
CN Cetemican
CN Cevalin
CN Cevatine
CN Cevex
CN Cevimin
CN Cevital
CN Cevitamic acid
CN Cevitamin
CN Cevitan
CN Cevitex
CN Chewcee
CN Ciamin
CN Cipca
CN Citrovit
CN Colascor
ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for DISPLAY
FS STEREOSEARCH
DR 56533-05-2, 57304-74-2, 57606-40-3, 56172-55-5, 129940-97-2, 14536-17-5, 50976-75-5, 154170-90-8, 89924-69-6, 30208-61-8, 259133-78-3
MF C6 H8 O6
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DIPPR*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PDLCOM*, PHAR, PHARMASEARCH, PIRA, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, TULSA, ULIDAT, USAN, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

44047 REFERENCES IN FILE CA (1967 TO DATE)
1119 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
44107 REFERENCES IN FILE CAPLUS (1967 TO DATE)
12 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L8 ANSWER 3 OF 3 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 87308368 MEDLINE
DOCUMENT NUMBER: 87308368 PubMed ID: 3624305
TITLE: Differentiation of axon-related Schwann cells in vitro. I. Ascorbic acid regulates basal lamina assembly and myelin

formation.

AUTHOR: Eldridge C F; Bunge M B; Bunge R P; Wood P M

CONTRACT NUMBER: NS07071 (NINDS)
NS09923 (NINDS)
SOURCE: JOURNAL OF CELL BIOLOGY, (1987 Aug) 105 (2) 1023-34.
Journal code: HMV; 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198709
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19870929

AB Rat Schwann cells cultured with dorsal root ganglion neurons in a serum-free defined medium fail to ensheathe or myelinate axons or assemble basal laminae. Replacement of defined medium with medium that contains human placental serum (HPS) and chick embryo extract (EE) results in both basal lamina and myelin formation. In the present study, the individual effects of HPS and EE on basal lamina assembly and on myelin formation by Schwann cells cultured with neurons have been examined. Some batches of HPS were unable to promote myelin formation in the absence of EE, as assessed by quantitative evaluation of cultures stained with Sudan black; such HPS also failed to promote basal lamina assembly, as assessed by immunofluorescence using antibodies against laminin, type IV collagen, and heparan sulfate proteoglycan. The addition of EE or L-ascorbic acid with such HPS led to the formation of large quantities of myelin and to the assembly of basal laminae. Pretreatment of EE with ascorbic acid oxidase abolished the EE activity, whereas trypsin did not. Other batches of HPS were found to promote both basal lamina and myelin formation in the absence of either EE or ascorbic acid. Ascorbic acid oxidase treatment or dialysis of these batches of HPS abolished their ability to promote Schwann cell differentiation, whereas the subsequent addition of ascorbic acid restored that ability. Ascorbic acid in the absence of serum was relatively ineffective in promoting either basal lamina or myelin formation. Fetal bovine serum was as effective as HPS in allowing ascorbic acid (and several analogs but not other reducing agents) to manifest its ability to promote Schwann cell differentiation. We suggest that ascorbic acid promotes Schwann cell myelin formation by enabling the Schwann cell to assemble a basal lamina, which is required for complete differentiation.

09/744,384
Paper # 11 Attach

$%^STN;HighlightOn= ***;HighlightOff=*** ;
Trying 3106016892...Open

Welcome to STN International! Enter x:x
LOGINID:SSSPTA1805RXY
PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

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NEWS 1 Web Page URLs for STN Seminar Schedule - N. America
NEWS 2 Sep 17 IMSworld Pharmaceutical Company Directory name change to PHARMASEARCH
NEWS 3 Oct 09 Korean abstracts now included in Derwent World Patents Index
NEWS 4 Oct 09 Number of Derwent World Patents Index updates increased
NEWS 5 Oct 15 Calculated properties now in the REGISTRY/ZREGISTRY File
NEWS 6 Oct 22 Over 1 million reactions added to CASREACT
NEWS 7 Oct 22 DGENE GETSIM has been improved
NEWS 8 Oct 29 AAASD no longer available
NEWS 9 Nov 19 New Search Capabilities USPATFULL and USPAT2
NEWS 10 Nov 19 TOXCENTER(SM) - new toxicology file now available on STN
NEWS 11 Nov 29 COPPERLIT now available on STN
NEWS 12 Nov 29 DWPI revisions to NTIS and US Provisional Numbers
NEWS 13 Nov 30 Files VETU and VETB to have open access
NEWS 14 Dec 10 WPINDEX/WPIDS/WPIX New and Revised Manual Codes for 2002
NEWS 15 Dec 10 DGENE BLAST Homology Search
NEWS 16 Dec 17 WELDASEARCH now available on STN
NEWS 17 Dec 17 STANDARDS now available on STN
NEWS 18 Dec 17 New fields for DPCI
NEWS 19 Dec 19 CAS Roles modified
NEWS 20 Dec 19 1907-1946 data and page images added to CA and CAplus
NEWS 21 Jan 25 BLAST(R) searching in REGISTRY available in STN on the Web
NEWS 22 Jan 25 Searching with the P indicator for Preparations
NEWS 23 Jan 29 FSTA has been reloaded and moves to weekly updates
NEWS 24 Feb 01 DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS 25 Feb 19 Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 26 Mar 08 Gene Names now available in BIOSIS
NEWS 27 Mar 22 TOXLIT no longer available
NEWS 28 Mar 22 TRCTHERMO no longer available

NEWS EXPRESS February 1 CURRENT WINDOWS VERSION IS V6.0d, CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP), AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
NEWS HOURS STN Operating Hours Plus Help Desk Availability
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FILE 'HOME' ENTERED AT 14:08:56 ON 22 MAR 2002

=> file caplus medline
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 0.15 0.15

FILE 'CAPLUS' ENTERED AT 14:09:21 ON 22 MAR 2002
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
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FILE 'MEDLINE' ENTERED AT 14:09:21 ON 22 MAR 2002

=> s different?
L1 3488082 DIFFERENT?

=> s l1 (20n) (anti (2n) oxidant?)
L2 96 L1 (20N) (ANTI (2N) OXIDANT?)

=> s l2 (20n) (cell or cells)
L3 25 L2 (20N) (CELL OR CELLS)

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 19 DUP REM L3 (6 DUPLICATES REMOVED)

=> d l4 kwic 1-19

L4 ANSWER 1 OF 19 CAPLUS COPYRIGHT 2002 ACS
AB . . . formation and a decrease in the prodn. of pregnancy-specific hormones. Due to the role of oxygen free radicals in trophoblast ***cell*** ***differentiation*** , we investigated the role of the key ***anti*** - ***oxidant*** enzyme, copper/zinc superoxide dismutase, encoded by chromosome 21 in in vitro trophoblast ***differentiation*** . We first obsd. that overexpression of superoxide dismutase in normal cytotrophoblasts impaired syncytiotrophoblast formation. This was assocd. with a significant. . .

L4 ANSWER 2 OF 19 CAPLUS COPYRIGHT 2002 ACS
AB . . . the assocd. ascorbate redox ratio. It was concomitant with a significant decrease in dehydroascorbate reductase activity. Thus, the pro- and ***anti*** - ***oxidant*** systems are involved in response to fusaric acid treatment although ***differential*** response of H2O2 and its metab.-related enzymes between the whole leaf and ***cell*** culture assays was found.

L4 ANSWER 3 OF 19 MEDLINE
AB . . . to cisplatin. The elevation was significantly higher in sensitive cells (3.3-fold) than in resistant (1.6- to 1.7-fold) cells. Exposure of ***cells*** to oxidative stress generated by menadione also resulted in enzyme induction but only in cisplatin-sensitive ***cells*** . Addition of ***anti*** - ***oxidants*** had ***different*** effects on the 2 inductions: N-acetylcysteine blocked the induction of both cisplatin and menadione, whereas catalase and glutathione-ester blocked only. . .

L4 ANSWER 4 OF 19 MEDLINE
AB . . . similarity with other bacterial genes involved in pathogenesis in plants, as well as in animals. The X. fastidiosa genome encodes ***different*** classes of proteins directly or indirectly involved in ***cell*** - ***cell*** interactions, degradation of plant ***cell*** walls, iron homeostasis, ***anti*** - ***oxidant*** responses, synthesis of toxins, and regulation of pathogenicity. Neither genes encoding members of the type III protein secretion system nor. . .

L4 ANSWER 5 OF 19 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 1
AB . . . tau. In the present study, the authors undertook to det. whether ROS generation or tau hyperphosphorylation mediate .beta.A-induced apoptosis. The ***anti*** - ***oxidant*** vitamin E or the kinase inhibitor N-(6-aminohexyl)-5-chloro-1-naphthalenesulfonamide (W7) was added following brief treatment of ***differentiated*** SH-SY-5Y human neuroblastoma ***cells*** with 22 .mu.M .beta.A. Under these conditions, vitamin E prevented ROS generation and apoptosis, but did not prevent intracellular calcium. . .

L4 ANSWER 6 OF 19 MEDLINE
AB . . . the MDS samples. This study shows that ineffective hemopoiesis in MDS could benefit from both NAC and ATRA, suggesting that ***anti*** - ***oxidant*** treatment may play a role in guaranteeing MDS ***cell*** survival, predisposing them towards ***differentiation*** .

L4 ANSWER 7 OF 19 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 2
AB . . . commensurate with the early phases of apoptosis, this increase did not occur in Bcl-2-expressing cells. Thus, Bcl-2 appears to allow ***cells*** to adapt to an increased state of oxidative stress, fortifying the cellular ***anti*** - ***oxidant*** defenses and counteracting the radical overprodn. imposed by ***different*** ***cell*** death stimuli. Furthermore, we report altered cytol. features of mitochondria during the early phases of apoptosis induced by C6-ceramide and. . .

L4 ANSWER 8 OF 19 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3
AB Peroxiredoxins (Prxs) are a newly defined family of ***anti*** - ***oxidant*** proteins that have been implicated, via their ***anti*** - ***oxidant*** activity, in a no. of cellular functions, including ***cell*** proliferation and ***differentiation*** , protection of other proteins from oxidative damage, and intracellular signaling. We isolated genomic DNA sequences of the Prx I genes. . .

L4 ANSWER 9 OF 19 CAPLUS COPYRIGHT 2002 ACS
AB In this presentation, the ability of ***different*** phenolic compds. to act as electron donors/acceptors will be discussed in conjunction with their physiol. ***anti*** -/pro- ***oxidant*** roles in ***cells*** . Two major pathways by which one-electron free radical intermediates of oxidn. of phenolic compds., phenoxyl radicals, are generated in cells. . .

L4 ANSWER 10 OF 19 MEDLINE
AB . . . with that of most patients with other neurological diseases. Such

elevations of HNE were sufficient to kill cyclic adenosine monophosphate (cAMP)- ***differentiated*** motor neuron hybrid ***cells*** in vitro, and ***anti*** - ***oxidants*** prevented this HNE-dependent ***cell*** death. These data suggest that oxidative stress and lipid peroxidation are associated with and may promote motor neuron degeneration in. . .

L4 ANSWER 11 OF 19 MEDLINE
AB . . . neurons were more vulnerable to the iron toxicity than calbindin (+) horizontal neurons. These findings show that iron exposure induces ***anti*** - ***oxidant*** -sensitive neuronal injury in retinal culture, independent of the excitotoxic or the apoptotic mechanisms. Of retinal neurons, ***different*** ***cell*** types exhibit differential vulnerabilities to the iron-induced oxidative injury. This simplified culture model system may be useful in elucidating mechanisms. . .

L4 ANSWER 12 OF 19 MEDLINE
AB . . . Retinoid action differs, in some respects, from other micronutrient anticancer mechanisms and appears to relate to its stimulation of cellular ***differentiation*** and resultant apoptosis of neoplastic ***cells*** . Combinations of ***anti*** - ***oxidant*** nutrients have been shown to be synergistic in their anticancer activity, probably due to their optimal anticancer activity at ***different*** oxygen potentials. Selectivity in the action on cancer ***cells*** , as opposed to normal ***cells*** , is a major feature of the ***anti*** - ***oxidant*** micronutrients.

L4 ANSWER 13 OF 19 MEDLINE
AB . . . suggests that PC12 sensitivity to PrP106-126 toxicity is related to prion protein expression and not to a state of high ***differentiation*** induced by NGF. Variants of PC12 ***cells*** that are more resistant to copper toxicity have higher levels of ***anti*** - ***oxidant*** enzymes, superoxide dismutase and glutathione peroxidase. Our results suggest that ***cells*** expressing higher levels of PrP(C) have higher resistance to oxidative stress or copper toxicity but are more sensitive to PrP106-126. . .

L4 ANSWER 14 OF 19 MEDLINE
AB . . . enhancing effect of incomplete Freund adjuvant (IFA) and polyoxyethylated castor oil upon the humoral immune response to sheep red blood ***cells*** (SRBC). None of the ***anti*** - ***oxidants*** tested inhibited the basal immune response to the antigen. In addition, mice inoculated with ***different*** concentrations of hydrogen peroxide showed an enhanced response against SRBC, mimicking the effect observed with adjuvants. Delayed type hypersensitivity induced. . .

L4 ANSWER 15 OF 19 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 4
TI Modifications of the ***anti*** - ***oxidant*** metabolism during proliferation and ***differentiation*** of colon tumor ***cell*** lines
AB ***Anti*** - ***oxidant*** metab. was studied at ***different*** times after sub-culture in 2 colon ***cell*** lines previously characterized for their growth and ***differentiation*** properties. The HT29 cell line is mainly composed of proliferating and undifferentiated cells, while the derived 5-fluorouracil (FUra)-adapted cells undergo. . .

L4 ANSWER 16 OF 19 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 5
AB Changes of the ***oxidant*** and ***anti*** - ***oxidant*** systems of alveolar macrophages under the influence of quartz were studied using ***different*** systems such as in vitro ***cell*** test, exptl. rat silicosis and patients with silicosis. The results showed that quartz dust could induce the prodn. of oxygen. . .

L4 ANSWER 17 OF 19 MEDLINE
AB . . . glutathione peroxidase (GPX, both Se-dependent and Se-independent), and glutathione reductase (GR) were measured in normal, nitrosoguanidine-transformed and SV40-transformed mouse liver ***cells*** in culture, as well as in mouse liver homogenates. Enzyme activities were compared on the basis of 3 ***different*** endpoints: per mg protein, per mg DNA, and per 10(6) ***cells*** . Except for GR, activity of all the measured ***anti*** - ***oxidant*** enzymes was much higher in vivo than in vitro. All of the anti-oxidant enzyme activities were lower in general in. . .

L4 ANSWER 18 OF 19 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 6
AB Oxidative phenomena are involved in human T-cell activation. The effect of ***different*** ***anti*** - ***oxidants*** (scavengers of O2-, OH, and lipo-oxygenase inhibitors) on the stimulation of murine T ***cells*** was studied. All the anti-oxidants used suppressed T-lymphocyte proliferation and IL-2 synthesis, the former effect resulting very likely from the. . .

L4 ANSWER 19 OF 19 MEDLINE
AB Butylated hydroxytoluene (BHT) which is widely used as an ***anti*** - ***oxidant*** in food has been found to induce the ***differentiation*** of murine erythroleukemia ***cells*** . BHT also amplifies the ***differentiation*** inducing activity of DMSO.

=> d l4 6, 8, 12-19 bib hit

L4 ANSWER 6 OF 19 MEDLINE
AN 2000118609 MEDLINE
DN 20118609 PubMed ID: 10654448
TI Efficacy of N-acetylcysteine and all-trans retinoic acid in restoring in vitro effective hemopoiesis in myelodysplastic syndromes.
CM Comment in: Leuk Res. 2000 Feb;24(2):139-40
AU Cortelezzi A; Cattaneo C; Sarina B; Cristiani S; Pomati M; Silvestris I; Motta M; Ibatici A; Gornati G; Volpe A D; Maiolo A T
CS Servizio Autonomo di Ematologia Diagnostica, Ospedale Maggiore Policlinico IRCCS, Milan, Italy.. cortelez@polic.cilea.it
SO LEUKEMIA RESEARCH, (2000 Feb) 24 (2) 129-37.
Journal code: K9M; 7706787. ISSN: 0145-2126.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200002
ED Entered STN: 20000229
Last Updated on STN: 20000229
Entered Medline: 20000211
AB We evaluated the in vitro effect on clonogenic potential (CFU-GM) and apoptosis in myelodysplastic syndromes (MDS) progenitors of an anti-oxidant (N-acetylcysteine, NAC) and/or a differentiating (all-trans retinoic acid, ATRA) agent. NAC significantly reduced apoptosis, both NAC and ATRA induced an increase in CFU-GM, but NAC seemed to be particularly effective in the high risk (HR) MDS. NAC + ATRA conferred a significant advantage in terms of CFU-GM with respect to NAC and ATRA alone. Tumor Necrosis Factor-alpha (TNF-alpha) levels decreased after incubation with NAC in the MDS samples. This study shows that ineffective hemopoiesis in MDS could benefit from both NAC and ATRA, suggesting that ***anti*** - ***oxidant*** treatment may play a role in guaranteeing MDS ***cell*** survival, predisposing them towards ***differentiation*** .

L4 ANSWER 8 OF 19 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3
AN 1999:700471 CAPLUS
DN 132:89010
TI Characterization of mouse peroxiredoxin I genomic DNA and its expression
AU Lee, T.-H.; Yu, S.-L.; Kim, S.-U.; Lee, K.-K.; Rhee, S. G.; Yu, D.-Y.
CS Yusong, P.O. Box 115, Korea Research Institute of Bioscience and Biotechnology, Taejon, S. Korea
SO Gene (1999), 239(2), 243-250
CODEN: GENED6; ISSN: 0378-1119
PB Elsevier Science B.V.
DT Journal
LA English
RE.CNT 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB Peroxiredoxins (Prxs) are a newly defined family of ***anti*** - ***oxidant*** proteins that have been implicated, via their ***anti*** - ***oxidant*** activity, in a no. of cellular functions, including ***cell*** proliferation and ***differentiation*** , protection of other proteins from oxidative damage, and intracellular signaling. We isolated genomic DNA sequences of the Prx I genes from the mouse, and characterized their mol. genetic features. Prx I was found to form a small gene family with two and three members; one functional and two pseudogenes. The Prx I-1 gene has splice donor/acceptor site sequences and five or six exons, whereas the Prx I-2 clone has several structural features characteristic of a typical retroposon found to have ORF sequences. We analyzed the expression of pseudogenes, which were not expressed on the transcription levels in the investigated organs. The functional copy of the Prx I-1 gene was expressed abundantly in liver and kidney of the adult, as well as in early developing embryos. This report, together with amino acid/nucleotide sequence similarity between human and mice, provides a basis for speculating on an even earlier event in the evolution of the Prx I gene family, i.e. the Prx I gene was well conserved in human and mice via its anti-oxidant activity.

Nov. 1, 1999 BSW

L4 ANSWER 12 OF 19 MEDLINE
AN 1998323701 MEDLINE
DN 98323701 PubMed ID: 9659516
TI Mechanisms of cancer inhibition by anti-oxidant nutrients.
AU Shklar G
CS Department of Oral Medicine and Diagnostic Sciences, Harvard School of Dental Medicine, Boston, MA 02115, USA.
SO ORAL ONCOLOGY, (1998 Jan) 34 (1) 24-9. Ref: 66
Journal code: CU5; 9709118. ISSN: 1368-8375.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 199807
ED Entered STN: 19980731

Last Updated on STN: 19980731
Entered Medline: 19980723

AB The cancer inhibitory properties of anti-oxidant micronutrients have been well established in experimental animal models and cell culture studies. Human studies have also tended to indicate an inhibition of various forms of cancer and the regression of some precancerous lesions. The biological mechanisms for cancer inhibition and regression are now gradually becoming understood, and the anti-oxidant nutrients appear to act through a number of pathways common to most of the agents studied. These various micronutrients appear to act through a complex group of "common pathways"
of anticancer activity based upon three major mechanisms: (1) tumour inhibition by immune cytokines; (2) stimulation of cancer suppressor genes, such as "wild type" p53, and diminished expression or dysregulation of oncogenes such as mutant p53 and H-ras; (3) inhibition of tumour angiogenesis through the inhibition of angiogenesis-stimulating factors such as TGF alpha. Retinoid action differs, in some respects, from other micronutrient anticancer mechanisms and appears to relate to its stimulation of cellular ***differentiation*** and resultant apoptosis of neoplastic ***cells*** . Combinations of ***anti*** - ***oxidant*** nutrients have been shown to be synergistic in their anticancer activity, probably due to their optimal anticancer activity at ***different*** oxygen potentials. Selectivity in the action on cancer ***cells*** , as opposed to normal ***cells*** , is a major feature of the ***anti*** - ***oxidant*** micronutrients.

L4 ANSWER 13 OF 19 MEDLINE
AN 1998303229 MEDLINE
DN 98303229 PubMed ID: 9641527
TI Effects of oxidative stress on prion protein expression in PC12 cells.
AU Brown D R; Schmidt B; Kretzschmar H A
CS Institut fur Neuropathologie, Universitat Gottingen, Germany.. drb33@cam.ac.uk
SO INTERNATIONAL JOURNAL OF DEVELOPMENTAL NEUROSCIENCE, (1997 Dec) 15 (8)
961-72.
Journal code: 126; 8401784. ISSN: 0736-5748.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199808
ED Entered STN: 19980828
Last Updated on STN: 19980828
Entered Medline: 19980820

AB PC12 cells are known to express the prion protein, a normal cell surface glycoprotein. This protein is upregulated in PC12 cells differentiated with nerve growth factor. A neurotoxic prion protein peptide, PrP106-126, is not toxic to PC12 cells alone. PrP106-126 is toxic to PC12 cells co-cultured with microglia and more so to NGF-differentiated PC12 cells. PC12 cells selected for resistance to either copper toxicity or oxidative stress have higher levels of PrP(C) expression. Both PC12 variants are more sensitive to the toxicity of PrP106-126. This suggests that PC12 sensitivity to PrP106-126 toxicity is related to prion protein expression and not to a state of high ***differentiation*** induced by NGF. Variants of PC12 ***cells*** that are more resistant to copper toxicity have higher levels of ***anti*** - ***oxidant*** enzymes, superoxide dismutase and glutathione peroxidase. Our results suggest that ***cells*** expressing higher levels of PrP(C) have higher resistance to oxidative stress or copper toxicity but are more sensitive to PrP106-126 toxicity. Prion protein expression may be involved in both the metabolism of copper and resistance to oxidative stress. Increased cellular resistance to copper toxicity may be partly related to increased activity of anti-oxidant enzymes.

L4 ANSWER 14 OF 19 MEDLINE
AN 96249508 MEDLINE
DN 96249508 PubMed ID: 8668921
TI Anti-oxidants inhibit the enhancement of the immune response caused by oil adjuvants.
AU Di Gianni P; Minnucci F S; Alves Rosa M F; Vulcano M; Isturiz M A
CS Instituto de Investigaciones Hematologicas, Academia Nacional de Medicina,
Buenos Aires, Argentina.
SO SCANDINAVIAN JOURNAL OF IMMUNOLOGY, (1996 Apr) 43 (4) 413-20.
Journal code: UCW; 0323767. ISSN: 0300-9475.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199608
ED Entered STN: 19960819
Last Updated on STN: 19980206
Entered Medline: 19960805

AB Adjuvants are agents that can induce strong immunity to different antigens. They are thought to act mainly by stimulating macrophages, causing the release of cytokines, which in turn induce an inflammatory focus necessary for the adjuvant action. The authors found that catalase, ascorbic acid, N-acetylcysteine and glutathione are able to inhibit the enhancing effect of incomplete Freund adjuvant (IFA) and polyoxyethylated castor oil upon the humoral immune response to sheep red blood ***cells*** (SRBC). None of the ***anti*** - ***oxidants*** tested inhibited the basal immune response to the antigen. In addition, mice inoculated with ***different*** concentrations of hydrogen peroxide showed an enhanced response against SRBC, mimicking the effect observed with adjuvants. Delayed type hypersensitivity induced by SRBC in the presence of IFA was also inhibited by catalase. In conclusion, the report indicates that oxygen radicals are crucial molecules involved in the adjuvant effect observed in SRBC immunized mice.

L4 ANSWER 15 OF 19 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 4
AN 1995:341388 CAPLUS
DN 122:129454
TI Modifications of the ***anti*** - ***oxidant*** metabolism during proliferation and ***differentiation*** of colon tumor ***cell*** lines
AU Bravard, A.; Beaumatin, J.; Dussaulx, E.; Lesuffleur, T.; Zweibaum, A.; Luccioni, C.
CS DSV, CEA, Fontenay-aux-Roses, 92265, Fr.
SO Int. J. Cancer (1994), 59(6), 843-7
CODEN: IJCNAW; ISSN: 0020-7136
DT Journal
LA English
TI Modifications of the ***anti*** - ***oxidant*** metabolism during proliferation and ***differentiation*** of colon tumor ***cell*** lines

AB ***Anti*** - ***oxidant*** metab. was studied at ***different*** times after sub-culture in 2 colon ***cell*** lines previously characterized for their growth and ***differentiation*** properties. The HT29 cell line is mainly composed of proliferating and undifferentiated cells, while the derived 5-fluorouracil (FUra)-adapted cells undergo growth-dependent differentiation, which is complete at post-confluence. In the 2 cell lines, all the anti-oxidant parameters studied appeared to be related to proliferation, with increased activity of superoxide dismutase (SOD) 1 and 2, catalase (CAT), glutathione peroxidase (GPX), glutathione reductase (GSR), and glutathione transferase (GST), and decreased glucose-6-phosphate dehydrogenase (G6PD) activity and
glutathione content, in parallel with slowing down of proliferation. At post-confluence, these metabolic parameters remained stable, except for GPX activity, which continued to increase, and CAT activity, which decreased. The amts. of SOD1, SOD2 and CAT immunoreactive proteins, estd.
by Western blotting, appeared to be correlated to their resp. enzymic activities. SOD1, CAT and GST activity and glutathione content, which remained at similar levels in the 2 cell lines for all times studied, appeared unrelated to the differentiation process. GSR and GPX activity, which was lower in FUra-adapted than in parental cells only at post-confluence, could be considered as markers of differentiated cells. The higher SOD2 and lower G6PD activity obsd. in FUra-resistant cell in comparison with parental cells at all times after sub-culture could be characteristic both of differentiative and of differentiated cells. Interestingly, cytogenetics have previously indicated that deletions of the long arm of chromosome 6, which carry the gene for SOD2, were frequently obsd. in parental but not in FUra-adapted cells. These results demonstrate that modifications of the anti-oxidant metab. occur in relation with proliferation and differentiation, and suggest a particular role for SOD2 in these cellular processes.

L4 ANSWER 16 OF 19 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 5
AN 1993:514632 CAPLUS
DN 119:114632
TI Oxidative injury of alveolar macrophage in the development of silicosis
AU Liu, Baohian; Zhang, Guogao; Guo, Ding; Yao, Rulin
CS Shanxi Med. Coll., Taiyuan, 030001, Peop. Rep. China
SO Zhonghua Yufang Yixue Zazhi (1993), 27(1), 10-12
CODEN: CHYCDW; ISSN: 0253-9624
DT Journal
LA Chinese

AB Changes of the ***oxidant*** and ***anti*** - ***oxidant*** systems of alveolar macrophages under the influence of quartz were studied using ***different*** systems such as in vitro ***cell*** test, exptl. rat silicosis and patients with silicosis. The results showed that quartz dust could induce the prodn. of oxygen derived free radicals in alveolar macrophages which could lead to lipid peroxidn. of cell membrane system. These changes were esp. obvious in the early stage of exptl. rat silicosis and in the patients with silicosis. The process of dust recycling involves ingestion, destruction and re-releasing of the dust by the alveolar macrophages, and was an important link in the prodn. and development of lung fibrosis. It also illustrated the role played by oxidative injury of alveolar macrophages in the development of lung fibrosis.

L4 ANSWER 17 OF 19 MEDLINE
AN 90109384 MEDLINE
DN 90109384 PubMed ID: 2558077
TI Antioxidant enzyme activities in normal and transformed mouse liver cells.
AU Sun Y; Oberley L W; Elwell J H; Sierra-Rivera E
CS Radiation Research Laboratory, University of Iowa, Iowa City 52242.
SO INTERNATIONAL JOURNAL OF CANCER, (1989 Dec 15) 44 (6) 1028-33.

Journal code: GQU; 0042124. ISSN: 0020-7136.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199002
ED Entered STN: 19900328
Last Updated on STN: 19900328
Entered Medline: 19900216
AB Copper- and zinc-containing superoxide dismutase (CuZnSOD), manganese-containing superoxide dismutase (MnSOD), catalase (CAT), glutathione peroxidase (GPX, both Se-dependent and Se-independent), and glutathione reductase (GR) were measured in normal, nitrosoguanidine-transformed and SV40-transformed mouse liver ***cells*** in culture, as well as in mouse liver homogenates. Enzyme activities were compared on the basis of 3 ***different*** endpoints: per mg protein, per mg DNA, and per 10(6) ***cells*** . Except for GR, activity of all the measured ***anti*** - ***oxidant*** enzymes was much higher in vivo than in vitro. All of the anti-oxidant enzyme activities were lower in general in the 2 transformed cell lines than in the in vitro normal cell line, except Cu-ZnSOD, which showed little change. However, MnSOD was the only enzyme
which showed lowered activity in both transformed cell lines, no matter what endpoint was used. This finding is in agreement with previous work showing lowered MnSOD activity in tumor cells.

L4 ANSWER 18 OF 19 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 6
AN 1990:34305 CAPLUS
DN 112:34305
TI Inhibition of murine T-cell responses by anti-oxidants: the targets of lipo-oxygenase pathway inhibitors
AU Dornand, J.; Gerber, M.
CS Lab. Biochim. Membr., CNRS, Montpellier, 34060, Fr.
SO Immunology (1989), 68(3), 384-91
CODEN: IMMUAM; ISSN: 0019-2805
DT Journal
LA English
AB Oxidative phenomena are involved in human T-cell activation. The effect of ***different*** ***anti*** - ***oxidants*** (scavengers of O2-, OH, and lipo-oxygenase inhibitors) on the stimulation of murine T ***cells*** was studied. All the anti-oxidants used suppressed T-lymphocyte proliferation and IL-2 synthesis, the former effect resulting very likely from the latter. This inhibition was concomitant with the triggering of activation. The various anti-oxidants have different biochem. targets. Unlike the other compds., the phenolic drugs nordihydroguaiaretic acid (NDGA) and butylated hydroxyanisole (BHA), which
block peroxidn., affect both signals triggered by the binding of lectin to its receptors: they suppress the rise of intracellular free Ca concn. and inhibit some of the events, depending on the sole protein kinase C activation, namely IL-2 receptor expression and phorbol myristate acetate-induced pH change. The results are discussed within the framework of a possible involvement of reactive O species and of arachidonic acid deriv.(s) in T-cell activation and IL-2 prodn.

L4 ANSWER 19 OF 19 MEDLINE
AN 84108894 MEDLINE
DN 84108894 PubMed ID: 6581988
TI Differentiation induction of murine erythroleukemia cells by butylated hydroxytoluene.
AU Ohno Y; Takuma T; Asahi K; Isono K
SO FEBS LETTERS, (1984 Jan 9) 165 (2) 277-9.
Journal code: EUH; 0155157. ISSN: 0014-5793.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198403
ED Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19840305
AB Butylated hydroxytoluene (BHT) which is widely used as an ***anti*** - ***oxidant*** in food has been found to induce the ***differentiation*** of murine erythroleukemia ***cells*** . BHT also amplifies the ***differentiation*** inducing activity of DMSO.

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